| Ref # | Hits | Search Query | DBs | Default Operator | Plurals | Time Stamp |
|---|---|---|---|---|---|---|
| S1 | 1 | ("6605442").PN. | USPAT | OR | OFF | 2005/05/06 12:12 |
| S2 | 0 | isgf3 with label | US-PGPUB; USPAT | ADJ | ON | 2005/05/06 11:56 |
| S3 | 112 | isgf3 | US-PGPUB; USPAT | ADJ | ON | 2005/05/06 11:56 |
| S4 | 0 | receptor recognition factor with label? | US-PGPUB; USPAT | ADJ | ON | 2005/05/06 12:13 |
| S5 | 26 | receptor recognition factor with label$ | US-PGPUB; USPAT | ADJ | ON | 2005/05/06 12:13 |

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                   (ROSPATENT) added to list of core patent offices covered
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                   data from INPADOC
 NEWS  5  FEB 28   BABS - Current-awareness alerts (SDIs) available
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 NEWS  9  MAR 03   MEDLINE file segment of TOXCENTER reloaded
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 NEWS  11 MAR 22   Original IDE display format returns to REGISTRY/ZREGISTRY
 NEWS  12 MAR 22   PATDPASPC - New patent database available
 NEWS  13 MAR 22   REGISTRY/ZREGISTRY enhanced with experimental property tags
 NEWS  14 APR 04   EPFULL enhanced with additional patent information and new
                   fields
 NEWS  15 APR 04   EMBASE - Database reloaded and enhanced
 NEWS  16 APR 18   New CAS Information Use Policies available online
 NEWS  17 APR 25   Patent searching, including current-awareness alerts (SDIs),
                   based on application date in CA/CAplus and USPATFULL/USPAT2
                   may be affected by a change in filing date for U.S.
                   applications.
 NEWS  18 APR 28   Improved searching of U.S. Patent Classifications for
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               AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

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=> index biosci
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
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                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.21       0.21

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       AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS,
       BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB,
       CROPU, DDFB, DDFU, DGENE, DISSABS, ...' ENTERED AT 12:02:33 ON 06 MAY
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=> s isgf3 (15A) label?
          1   FILE BIOSIS
          2   FILE BIOTECHNO
          3   FILE CANCERLIT
          1   FILE CAPLUS
          1   FILE DRUGU
  31 FILES SEARCHED...
          1   FILE EMBASE
          1   FILE ESBIOBASE
          2   FILE LIFESCI
          1   FILE MEDLINE
          2   FILE SCISEARCH
          2   FILE USPATFULL
  71 FILES SEARCHED...

  11 FILES HAVE ONE OR MORE ANSWERS,   75 FILES SEARCHED IN STNINDEX

L1   QUE ISGF3 (15A) LABEL?

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                                                         ENTRY    SESSION
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FILE 'MEDLINE' ENTERED AT 12:03:37 ON 06 MAY 2005

=> s l1
L2 17 L1

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3 6 DUP REM L2 (11 DUPLICATES REMOVED)

=> d l3 bib ab 1-6

L3 ANSWER 1 OF 6 USPATFULL on STN
AN 2004:267774 USPATFULL
TI Methods to identify agents that increase or decrease UBP43 activity and methods for use of such agents
IN Zhang, Dong-Er, San Diego, CA, UNITED STATES
Yan, Ming, San Diego, CA, UNITED STATES
Malakhova, Oxana A., San Diego, CA, UNITED STATES
PI US 2004209315 A1 20041021
AI US 2004-771951 A1 20040203 (10)
PRAI US 2003-444941P 20030203 (60)
DT Utility
FS APPLICATION
LREP BAKER & BOTTS, 30 ROCKEFELLER PLAZA, NEW YORK, NY, 10112
CLMN Number of Claims: 54
ECL Exemplary Claim: 1
DRWN 5 Drawing Page(s)
LN.CNT 1878
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to identification of agents that modulate UBP43 activity as well as associated methods, uses, processes, compositions and agents. In particular, the invention is directed to in vivo and in vitro methods to identify an agent that inhibits or stimulates UBP43 activity within a cell, a method for inducing cellular apoptosis, a method for affecting cellular reaction to interferon, a method for treating disease associated with cellular proliferation by causing apoptosis, and a method for treating both acute and chronic diseases in which interferon exerts a beneficial effect. The invention is also directed to modified ISG15-conjugates that have lowered or no susceptibility to UBP43 cleavage, pharmaceutical compositions of the agents, conjugates, and additional modified ISG15-conjugates.

L3 ANSWER 2 OF 6 USPATFULL on STN
AN 2001:152761 USPATFULL
TI Accessory factory function for interferon gamma and its receptor
IN Pestka, Sidney, North Caldwell, NJ, United States
Kotenko, Serguei, Highland Park, NJ, United States
Soh, Jaemog, Highland Park, NJ, United States
Donnelly, Robert J., Highland Park, NJ, United States
Mariano, Thomas M., Somerset, NJ, United States
Cook, Jeffry R., Kendall Park, NJ, United States
Emanuel, Stuart, New Brunswick, NJ, United States
Schwartz, Barbara, Annandale, NJ, United States
PA University of Medicine & Dentistry of New Jersey, Newark, NJ, United States (U.S. corporation)
PI US 6287853 B1 20010911
AI US 1997-871572 19970609 (8)
RLI Continuation of Ser. No. US 1995-444134, filed on 18 May 1995, now abandoned Division of Ser. No. US 1993-110119, filed on 20 Aug 1993, now abandoned
DT Utility
FS GRANTED
EXNAM Primary Examiner: Saoud, Christine J.
LREP Muccino, Richard R.
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 32 Drawing Figure(s); 24 Drawing Page(s)
LN.CNT 3188
AB This invention relates (a) to a 540 kb YAC which encodes the necessary species-specific factor(s) and is able to substitute for human Chromosome 21 to reconstitute the Hu-IFN-gamma receptor-mediated induction of class I HLA antigens; (b) to the construction of a plasmid to integrate the selective marker for antibiotic G418 resistance into YACs and to delete some of the human DNA fragments from YACs in order to facilitate the manipulation of human genomic DNA in yeast artificial chromosome (YAC) clones; (c) to two fragmentation vectors, pSE1 and pSE2, which contain a neomycin resistance and URA3 gene, developed for targeting yeast artificial chromosomes (YACs) containing human genomic DNA; (d) to a chromosomal fragmentation procedure employed to produce a deletion set of yeast artificial chromosomes (YACs) from a parental YAC (GART D142H8) known to map to Chromosome 21q and to encode the human interferon-gamma receptor (Hu-IFN-gamma R) accessory factor gene as well as the phosphoribosylglycinamide formyltranisferase (GART) gene; and (e) to the isolation of cDNA clones that encode the necessary species-specific factor and that are able to substitute for human Chromosome 21 to reconstitute the Hu-IFN-gamma receptor-mediated induction of class I HLA antigens.

L3 ANSWER 3 OF 6 CANCERLIT on STN DUPLICATE 1
AN 97413783 CANCERLIT

DN 97413783 PubMed ID: 9268319
TI Regulation of interferon-alpha responsiveness by the duration of Janus kinase activity.
AU Lee C K; Bluyssen H A; Levy D E
CS Department of Pathology and Kaplan Cancer Center, New York University School of Medicine, New York, New York 10016, USA.
NC AI28900 (NIAID)
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1997 Aug 29) 272 (35) 21872-7. Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS MEDLINE; Priority Journals
OS MEDLINE 97413783
EM 199710
ED Entered STN: 19971105
Last Updated on STN: 20021018
AB Daudi B lymphoblastoid cells are highly sensitive to the anti-growth and anti-viral effects of interferon (IFN). Unlike many cell lines, these cells show prolonged transcription of IFN-stimulated genes following treatment with IFN-alpha. This prolonged response correlated with the continued presence of the activated transcription factor, IFN-stimulated gene factor 3 ( ***ISGF3***. ). Pulse-chase ***labeling*** experiments indicated that the half-life of the phosphorylation of signal transducers and activators of transcription (Stat)1 and Stat2 was short (<2 h) although the turnover of the proteins themselves was slow (>24 h), indicative of a constitutive phosphatase activity. The administration of protein-tyrosine kinase inhibitors at any time point during IFN stimulation led to rapid inhibition of the response, indicating that tyrosine kinase activity was continuously required. Catalytic activity of Jak1 and Tyk2 kinases remained elevated for prolonged periods following stimulation. Continuous presence of IFN-alpha was necessary for maintaining prolonged activation of ISGF3 and of Janus kinases, an activity that was blocked by antibodies to IFN-alpha or by cycloheximide. Conditioned medium of IFN-alpha-stimulated cells was capable of stimulating STAT activation in naive cells. Taken together, these results suggest that the response to IFN-alpha is controlled by the duration of stimulated Janus kinase activity over the background of constitutive dephosphorylation and that this response can be sustained by autocrine secretion of IFN-alpha.

L3 ANSWER 4 OF 6 CANCERLIT on STN
AN 1998637782 CANCERLIT
DN 98637782
TI Interferon-alpha resistance in a cutaneous T cell lymphoma cell line is associated with loss of the STAT1 protein (Meeting abstract).
AU Sun W H; Jandeska S; Pabon C; Rosen S T
CS Lurie Cancer Center, Northwestern University Medical School, Chicago, IL 60614.
SO Proc Annu Meet Am Assoc Cancer Res, (1997) 38 A782. ISSN: 0197-016X.
DT (MEETING ABSTRACTS)
LA English
FS Institute for Cell and Developmental Biology
EM 199801
ED Entered STN: 19980109
Last Updated on STN: 19980109
AB Cutaneous T cell lymphoma (CTCL) is characterized by a clonal malignant proliferation of mature helper T cells in the skin with ultimate progression involving lymph nodes, peripheral blood and viscera. Administration of recombinant interferon alpha-2a (IFNalpha-2a) has been shown to be one of the most effective therapies for CTCL. However, the efficacy of IFNalpha-2a is limited by the development of resistance in some patients who received continuous therapy. IFNalpha belongs to the Type-I IFN family and binds to the Type-I IFN receptor (IFNR). Phosphorylation of IFNR, immediately after ligand binding, is regulated by two Janus kinases (Tyk-2 and Jak-1). Tyk-2 and Jak-1 themselves also become phosphorylated in cells upon IFNalpha stimulation. The activated Tyk-2 and Jak-1 then induce phosphorylation of interferon-regulated signal transducers and activators of transcription (STATs). Activated STAT 1 and 2 can associate with a 48 kD protein (p48) to form the interferon-stimulated gene factor-3 (ISGF-3) complex which binds specifically to the IFNalpha-stimulated response element (ISRE), resulting in gene transcription. More recently, STAT3 was reported to be phosphorylated upon IFNalpha treatment and form a protein-DNA complex, distinct from the ISGF3 complex. We have developed an IFN resistant CTCL cell line (HUT78R) by culturing the IFN-sensitive cells (HUT78S) in increasing concentration of IFNalpha-2a (up to 1 x 10(6) U/ml) for a prolonged period. The levels of IFNR mRNA expression were found to be comparable between the two lines, by Northern and Slot blot analyses. The HUT78R and S lines also exhibited similar levels of binding sites and binding affinity for 125I-labeled recombinant IFNalpha-2a determined by Scatchard analysis. By gel shift analysis, we found that IFNalpha induced the ***ISGF3*** complex formation using the ***labeled*** ISRE probe and this DNA-protein interaction was inhibited in the HUT78R cells. We then examined STAT protein activation in HUT78 cells and our results showed that phosphorylation of STAT1 was completely inhibited in the resistant cells. However, IFNalpha-induced STAT2 phosphorylation was comparable between the HUT78R and HUT78S lines. Both lines exhibited a low level of constitutive STAT3 phosphorylation and an increased level of STAT3 phosphorylation can be induced upon IFNalpha-2a treatment. To our surprise, we did not detect any STAT1 (alpha and beta) protein in the HUT78R cells by immunoblotting analysis. RT-PCR results revealed that both cell lines contain the STAT1 transcript, using primers encoding the first five exons. However, it is not clear if there are mutation(s) further downstream that may cause premature termination of the transcript. We are currently investigating these possibilities. In summary, our findings suggest that IFNalpha-resistance are caused by the loss of STAT1 protein in a human cancer cell line.

L3 ANSWER 5 OF 6 DRUGU COPYRIGHT 2005 THE THOMSON CORP on STN
AN 1995-33339 DRUGU C M
TI Interferon receptor recognition peptides enhance the biological potency of interferon alphas.
AU Fish E n
CS Univ.Toronto
LO Toronto, Ont., Can.
SO FEBS Lett. (365, No. 1, 87-91, 1995) 4 Fig. 25 Ref.
CODEN: FEBLAL ISSN: 0014-5793
AV Department of Microbiology, University of Toronto, FitzGerald Bldg., 150 College Street, Toronto, Ont., M5S 1A8, Canada.
LA English
DT Journal
FA AB; LA; CT

FS Literature
AB 3 Peptides were prepared which corresponded to putative receptor recognition domains of IFN; they were designated IFN receptor recognition peptides (IRRP): CLKDRHD (IRRP-1), ESLLEKFYTELYQQLND (IRRP-2) and YFQRITLYLTEKKYSPCA (IRRP-3). The peptides increased the extent of 125I-IFN-alpha-2 binding to Daudi cells, and enhanced the activation of the transcription factor ISGF3 induced by IFN-Con-1 (a consensus IFN-alpha, Amgen) in Daudi and MRC-5 cells. Human glial T98G cells were challenged with EMC virus (EMCV); the peptides enhanced the antiviral activity of suboptimal doses of IFN-Con-1 vs. EMCV. However, the IRRP peptides had little ability to augment the antiproliferative action of higher doses of IFN-Con-1 vs. T98G cells.

L3 ANSWER 6 OF 6 CANCERLIT on STN DUPLICATE 2
AN 92346719 CANCERLIT
DN 92346719 PubMed ID: 1638633
TI A transcription factor with SH2 and SH3 domains is directly activated by an interferon alpha-induced cytoplasmic protein tyrosine kinase(s).
AU Fu X Y
CS Department of Biochemistry, Mount Sinai School of Medicine, New York, New York 10029.
SO CELL, (1992 Jul 24) 70 (2) 323-35.
Journal code: 0413066. ISSN: 0092-8674.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS MEDLINE; Priority Journals
OS MEDLINE 92346719
EM 199209
ED Entered STN: 19990618
Last Updated on STN: 19990618
AB Interferon-stimulated gene factor 3 (ISGF3), the primary transcription factor induced by interferon alpha, is a complex of four (113, 91, 84, and 48 kd) proteins. This paper reports that the 113, 91, and 84 kd (ISGF3 alpha) proteins of ISGF3 contain conserved SH2 and SH3 domains. A specific interferon alpha-induced cytoplasmic protein tyrosine kinase(s) can form a transient complex with ISGF3 alpha proteins. These ISGF3 alpha proteins can be immunoprecipitated by anti-phosphotyrosine antibodies only after interferon alpha treatment. Phosphoamino acid analyses of 32P-***labeled*** ***ISGF3*** alpha proteins confirm that ***ISGF3*** alpha proteins are directly tyrosine phosphorylated both in vitro and in vivo in response to interferon alpha, and this tyrosine phosphorylation can be inhibited by staurosporine and genistein. Phosphatase treatment of these ISGF3 alpha proteins results in inhibition of ISGF3 complex formation in vitro. These observations indicate that interferon alpha-induced direct tyrosine phosphorylation of ISGF3 alpha proteins is necessary for activation of the transcription factor ISGF3.

=> d his

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INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, ...' ENTERED AT 12:02:33 ON 06 MAY 2005

SEA ISGF3 (15A) LABEL?
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1 FILE BIOSIS
2 FILE BIOTECHNO
3 FILE CANCERLIT
1 FILE CAPLUS
1 FILE DRUGU
1 FILE EMBASE
1 FILE ESBIOBASE
2 FILE LIFESCI
1 FILE MEDLINE
2 FILE SCISEARCH
2 FILE USPATFULL
L1 QUE ISGF3 (15A) LABEL?
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L3 6 DUP REM L2 (11 DUPLICATES REMOVED)

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FULL ESTIMATED COST                              20.89      22.28

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      AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS,
      BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB,
      CROPU, DDFB, DDFU, DGENE, DISSABS, ...' ENTERED AT 12:09:08 ON 06 MAY
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          7   FILE BIOSIS
          2   FILE BIOTECHABS
          2   FILE BIOTECHDS
          1   FILE BIOTECHNO
          1   FILE CANCERLIT
          5   FILE CAPLUS
          3   FILE CEABA-VTB
          1   FILE CIN
  23 FILES SEARCHED...
         34   FILE DGENE
          1   FILE EMBASE
          1   FILE ESBIOBASE
  36 FILES SEARCHED...
         90   FILE GENBANK
          8   FILE IFIPAT
          1   FILE MEDLINE
          1   FILE SCISEARCH
          2   FILE TOXCENTER
         29   FILE USPATFULL
          1   FILE USPAT2
          3   FILE WPIDS
  73 FILES SEARCHED...
          3   FILE WPINDEX

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L4    QUE RECEPTOR (W) RECOGNITION (W) FACTOR

=> s l4 (15a) label?
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          3   FILE IFIPAT
  55 FILES SEARCHED...
         25   FILE USPATFULL
          1   FILE USPAT2

   3 FILES HAVE ONE OR MORE ANSWERS,    75 FILES SEARCHED IN STNINDEX

L5    QUE L4 (15A) LABEL?

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                                                 ENTRY    SESSION
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FILE 'USPAT2' ENTERED AT 12:11:19 ON 06 MAY 2005
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L6           29 L5

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7           25 DUP REM L6 (4 DUPLICATES REMOVED)

=> l7 not l3
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The previous command name entered was not recognized by the system.
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"HELP COMMANDS" at an arrow prompt (=>).

=> s l7 not l3
L8           25 L7 NOT L3

=> s l8 and pd<1993
L9            0 L8 AND PD<1993

=> d l8 bib ab 24-25

L8   ANSWER 24 OF 25  USPATFULL on STN
AN     1999:136984  USPATFULL
TI     Nucleic acids encoding receptor recognition factor Stat1.alpha. and
       Stat1.beta., and methods of use thereof
IN     Darnell, Jr., James E., Larchmont, NY, United States
       Schindler, Christian W., New York, NY, United States
       Fu, Xin-Yuan, Forrest Hills, NY, United States
       Wen, Zilong, New York, NY, United States
       Zhong, Zhong, New York, NY, United States
PA     The Rockefeller University, New York, NY, United States (U.S.
```

corporation)
PI US 5976835 19991102
AI US 1997-820754 19970319 (8)
RLI Division of Ser. No. US 1994-212185, filed on 11 Mar 1994 which is a continuation-in-part of Ser. No. US 1993-126588, filed on 24 Sep 1993, now abandoned And Ser. No. US 1993-126595, filed on 24 Sep 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-980498, filed on 23 Nov 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-854296, filed on 19 Mar 1992, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Spector, Lorraine
LREP Klauber & Jackson
CLMN Number of Claims: 36
ECL Exemplary Claim: 1
DRWN 53 Drawing Figure(s); 46 Drawing Page(s)
LN.CNT 4986
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Receptor recognition factors exist that recognizes the specific cell receptor to which a specific ligand has been bound, and that may thereby signal and/or initiate the binding of the transcription factor to the DNA site. The receptor recognition factor is in one instance, a part of a transcription factor, and also may interact with other transcription factors to cause them to activate and travel to the nucleus for DNA binding. The receptor recognition factor appears to be second-messenger-independent in its activity, as overt perturbations in second messenger concentrations are of no effect. The concept of the invention is illustrated by the results of studies conducted with interferon (IFN)-stimulated gene transcription, and particularly, the activation caused by both IFN.alpha. and IFN-.gamma.. Specific DNA and amino acid sequences for various human and murine receptor recognition factors are provided, as are polypeptide fragments of two of the ISGF-3 genes, and antibodies have also been prepared and tested. The polypeptides confirm direct involvement of tyrosine kinase in intracellular message transmission. Numerous diagnostic and therapeutic materials and utilities are also disclosed.

L8 ANSWER 25 OF 25 USPATFULL on STN
AN 1999:92527 USPATFULL
TI Mammalian ob polypeptides capable of modulating body weight, corresponding nucleic acids, and diagnostic and therapeutic uses thereof
IN Friedman, Jeffrey M., New York, NY, United States
Zhang, Yiying, New York, NY, United States
Proenca, Ricardo, Astoria, NY, United States
Maffei, Margherita, Asciano, Italy
Halaas, Jeffrey L., New York, NY, United States
Gajiwala, Ketan, New York, NY, United States
Burley, Stephen K., New York, NY, United States
PA The Rockefeller University, New York, NY, United States (U.S. corporation)
PI US 5935810 19990810
AI US 1994-347563 19941130 (8)
RLI Continuation-in-part of Ser. No. US 1994-292345, filed on 17 Aug 1994
DT Utility
FS Granted
EXNAM Primary Examiner: Railey, II, Johnny F.
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CLMN Number of Claims: 27
ECL Exemplary Claim: 1
DRWN 38 Drawing Figure(s); 35 Drawing Page(s)
LN.CNT 3413
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates generally to the control of body weight of animals including mammals and humans, and more particularly to materials identified herein as modulators of weight, and to the diagnostic and therapeutic uses to which such modulators may be put. In its broadest aspect, the present invention relates to the elucidation and discovery of nucleotide sequences, and proteins putatively expressed by such nucleotides or degenerate variations thereof, that demonstrate the ability to participate in the control of mammalian body weight. The nucleotide sequences in object represent the genes corresponding to the murine and human ob gene, that have been postulated to play a critical role in the regulation of body weight and adiposity. Preliminary data, presented herein, suggests that the polypeptide product of the gene in question functions as a hormone. The present invention further provides nucleic acid molecules for use as molecular probes, or as primers for polymerase chain reaction (PCR) amplification, i.e., synthetic or natural oligonucleotides. In further aspects, the present invention provides a cloning vector, which comprises the nucleic acids of the invention; and a bacterial, insect, or a mammalian expression vector, which comprises the nucleic acid molecules of the invention, operatively associated with an expression control sequence. Accordingly, the invention further relates to a bacterial or a mammalian cell transfected or transformed with an appropriate expression vector, and correspondingly, to the use of the above mentioned constructs in the preparation of the modulators of the invention. Also provided are antibodies to the ob polypeptide. Moreover, a method for modulating body weight of a mammal is provided. In specific examples, genes encoding two isoforms of both the murine and human ob polypeptides are provided.

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| COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION |
|---|---|---|
| FULL ESTIMATED COST | 8.25 | 32.89 |

SESSION WILL BE HELD FOR 60 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 12:12:56 ON 06 MAY 2005